



Letter

Salivary cotinine measurement for all children with persistent asthma: spit matters

Despite awareness that secondhand smoke (SHS) exacerbates asthma symptoms, more than half of US children with asthma are exposed to SHS,¹ and the parent is the primary source of exposure.^{2,3} Explanations for failure to prevent SHS exposure in the home for children with asthma are multifaceted and not well understood, and significant underreporting of SHS exposure occurs.⁴ Furthermore, few health care professionals use biomarkers (ie, cotinine) as an objective measure of SHS exposure.⁵ There has been minimal effort to examine the factors associated with persistent SHS exposure in high-risk children with asthma. This study aimed to investigate the pattern and factors associated with persistent high cotinine concentrations in a sample of urban children with asthma who had frequent emergency department (ED) visits.

Participants were 116 children aged 3 to 12 years with persistent asthma and high ED use. All were enrolled in a randomized clinical trial that examined the effectiveness of a behavioral environmental control intervention.⁶ The Johns Hopkins Medical Institutional Review Board approved the study, and written informed consent was obtained. Children were enrolled during an asthma ED visit and followed up for more than 12 months (baseline and 6 and 12 months). Inclusion criteria were physician-diagnosed persistent asthma, 2 or more ED asthma visits or 1 or more hospitalization in the past 12 months, and residence in a local metropolitan area. Salivary cotinine testing, an established biomarker of tobacco smoke absorption with a half-life of 16 to 24 hours, was conducted at each time point and analyzed using an enzyme immunoassay analysis at the Johns Hopkins Institute for Clinical and Translational Research. SHS exposure was defined as a cotinine concentration of 1.0 ng/mL or higher. Concurrently, caregivers reported household smoking behavior (ie, number and child relationship of every household smoker and type of home smoking restrictions [total, partial, none]).⁷ Other child environments with potential SHS exposure were ascertained. Caregiver stress and health care use measures, including number of asthma ED visits, hospitalizations, and primary care physician (PCP) visits, reported in the past 3 months were considered. Pharmacy records

were used to identify asthma medications dispensed in the prior 12 months.

Mean (SD) cotinine concentrations were examined across the 3 time points. Unadjusted analysis was limited to the 6-month time point because of a lack of difference in mean cotinine levels between 6 and 12 months. Cotinine status (positive vs negative) was compared using standard χ^2 tests and analysis of variance. Logistic regression models were tested to predict a positive cotinine test result at 6 months while adjusting for baseline cotinine status, number short-acting β -agonist (SABA) fills, number of routine primary care visits for asthma during the past 3 months, number of smokers in the home, and caregiver daily stress. Two-sided tests were used, and $P \leq .05$ was considered statistically significant.

Most children were male (63%), African American (95%), and enrolled in Medicaid (96%), with a mean (SD) age of 6.3 (2.7) years. Caregivers were single (75%), achieved an educational level of high school or more (81.9%), and were unemployed (44%), with a mean (SD) age of 31.4 (7.4) years. Caregiver daily stress was moderately high (mean [SD], 5.8 [2.9] of 10). Mean symptom days in past 2 weeks and nights during the past 4 weeks were high (5.7 of 14 days and 6.5 of 30 nights, respectively). Although 72% reported a total home smoking ban, more than half of children had a positive cotinine test result at each time point (baseline: 53 [54%] of 98, 6 months: 52 [57%] of 91, and 12 months: 51 [56%] of 91). Cotinine testing identified 17 (32%) of 53 children with a cotinine concentration of 1 ng/mL or higher but who had *no* history of a smoker in the home. There was some bidirectional change in cotinine status between baseline and 6 months (positive baseline change to negative: 31%; negative baseline change to positive: 25%). Mean cotinine concentrations significantly increased from baseline to 6 months ($P = .03$) but not between 6 and 12 months ($P = .96$) (Fig 1). Factors significantly associated with 6-month positive cotinine test result (unadjusted analysis) included a higher baseline cotinine level, higher mean controller and SABA fills, increased number of routine PCP and ED visits in the past 3 months, and increased number of household smokers. Maternal smoking, type of home smoking ban, caregiver stress, and season of cotinine collection were not associated with a positive cotinine test result at 6 months. Factors associated with a positive cotinine test result at 6 months in the final regression model were a positive baseline cotinine test result (odds ratio, 6.5; 95% CI, 2.3–9.2) and an increased number of PCP visits in the past 3 months (odds ratio, 1.85; 95% CI, 1.0–3.4) while controlling for number of SABA fills and smokers in the home.

This study confirms that SHS exposure is common among urban children with asthma,⁸ with relative fluctuation in SHS

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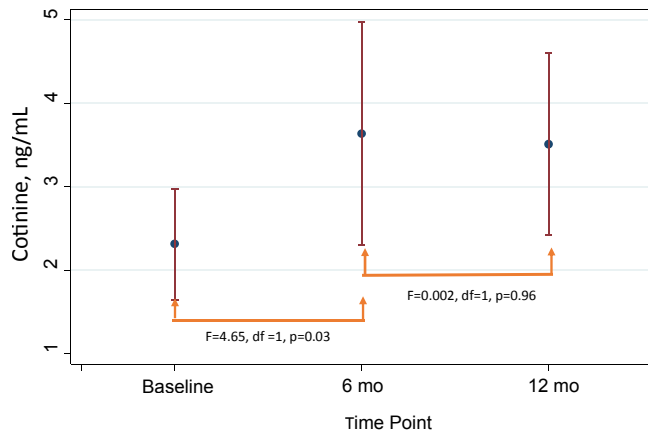


Figure 1. Mean cotinine levels in 12 months. Error bars indicate 95% confidence intervals.

exposure as evidenced by bidirectional change in cotinine levels at 6 months. Perhaps a low baseline (ED visit) cotinine level that significantly increased at 6 months reflects some effort to reduce SHS exposure when a child experiences acute asthma symptoms. However, caregivers appear challenged to change long-standing behaviors (ie, quit smoking and enforce a total home smoking ban). Prior reports indicate that child respiratory symptoms were not significantly associated with parental intention to quit smoking.⁹ Our findings indicate that children with a positive 6-month cotinine result had a mean of 1 PCP visit within the past 3 months, indicating recent health care use, perhaps a missed opportunity for assessing SHS exposure. Our data suggest that relying on self-report of SHS exposure will misclassify approximately one-third of children as being nonexposed. Although several commercial laboratories provide salivary cotinine testing, few PCPs order cotinine tests primarily because of a prior authorization requirement from some insurers and prohibitive patient cost (range, \$91–\$131 per test) (Laboratory Corporation of America [LabCorp] and Quest Diagnostics, personal oral communication, February 2, 2016). We advocate for a cotinine measurement of every child with persistent asthma during an acute or routine asthma care visit and promote expanding coverage by all insurers for cotinine testing in high-risk children with asthma. Study limitations include lack of accounting for all SHS exposures, long-term exposure reflected in a one-time salivary cotinine measurement, and generalizability reflected in the high-risk asthma inclusion criteria. Despite these limitations, the study suggests that using sequential salivary cotinine measurement reduces misclassification of children with asthma reportedly not exposed to SHS.

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